Modern Concepts of Cardiovascular Disease

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AORTIC STENOSIS

Interest in the clinical significance of aortic stenosis has been increasing during the past decade. Early confusions are becoming clarified. It is the purpose of this brief resume to emphasize certain present day accepted view points and to add a summary of a new series of 34 autopsied cases of our own.

Diagnosis

It seems clear that aortic stenosis is much more frequent than diagnosed, then and now. Factors explaining the infrequency of diagnosis are: the concept that aortic stenosis is a relatively rare disease; the misconception that a basal systolic *thrill* must be palpated; that the basal systolic murmur is due to an aortitis; that the systolic murmur is inaudible because of the accompanying louder diastolic murmur of aortic insufficiency.

A history of acute rheumatic fever may be frequently elicited or suspected. Cardiac symptoms may be completely absent,—the patient dying of an independent disease (Margolies et al)—or may appear late, (after 50), despite long preexisting pathology. "Heart consciousness," palpitation, dull precordial ache, faintness, vertigo, exertional dyspnea are among the early presenting complaints. However, the usually late episodes of angina pectoris, heart failure, syncope, sudden death, often unexpectedly usher in the clinical picture.

Angina pectoris is a frequent accompaniment, although not so closely connected with effort as in classical angina (Harrison), and may be overshadowed by the presence of distressing dyspnea. Earlier beliefs of the causal relationship of angina pectoris to aortic insufficiency, coronary arteriosclerosis or hypertension have given way to an ingenious explanation of myocardial ischemia, due to a suction action withdrawing blood from the coronary arteries because of the calcified aortic valves (Contratto and Levine). Narrowing of the mouths of the coronary arteries as a cause of angina has also been suggested (Boas).

Heart failure is usually of the left ventricular type, dyspnea, cardiac asthma, pulmonary engorgement, cough ("bronchitis") with bloody expectoration or frank hemoptysis, insomnia and weakness in the aged (Fishberg), being prominent symptoms. Vertigo, syncope and sudden death are frequent accompaniments of aortic stenosis (Marvin and Sull.van). The circulatory dynamics responsible for their occurrence are still not clear. Cerebral ischemia, a hyperactive carotid sinus, a mechanism similar to that which prevails in the sudden death of angina pectoris (Marvin and Sullivan) have all been suggested. In our own series to be commented on later, embolic episodes and coronary thrombosis were responsible for 4 cases of sudden death. Boas believes that sudden heart failure in aortic stenosis often simulates coronary thrombosis. Patients with aortic stenosis suffering with these curious unexplained attacks of loss of consciousness are the ones who frequently die instantly. Marvin points out that the fainting spells (syncope) usually occur on slight exertion—quiet walking about the house or along a level street—and last from 5 to 20 or 30 minutes—the longest 2 hours. The suggestion that a hyperactive carotid sinus is responsible for the syncopal episodes is an attractive hypothesis and is supported by considerable clinical and electrocardiographic evidence.

Physical Findings

A basal systolic murmur, somewhat rough and moderately pitched, submanubrial or in the aortic area, accompanied or not by a palpable systolic thrill is the most significant single physical finding. The systolic thrill which may be demonstrated usually late in the disease, probably coinciding with progressive calcification of the aortic cusps, is best felt with the patient in the upright posture, leaning slightly forward and at the height of *expiration*. The second aortic sound may be faint or absent, accompanied or replaced by a diastolic murmur, heard best along the left sternal border (aortic insufficiency). The heart may be only slightly increased to the left on percussion, the apex beat forceful and enduring due to concentric hypertrophy of the left ventricle. At autopsy the heart weight is usually markedly increased—particularly the left ventricle. The radial pulse is of the "plateau" type (pulsus tardus et parvus) and is often slow in rate (Contratto and Levine).

A significant, relatively recent addition to the objective diagnosis of calcific aortic stenosis is the roentgenological demonstration of calcified deposits in the aortic valves (Sosman and Wosika). While extensive calcified deposits may be seen in X-ray films, fluoroscopy of the heart, in either the right or left anterior oblique view is of greatest value in demonstrating these brightly appearing dancing shadows. Differentiation between calcification of the mitral valve must of course be made and may be difficult.

Rheumatic infection appears to be the most frequent cause of aortic stenosis (Christian and others). Subacute bacterial endocarditis may be superimposed. Aortic stenosis is seen both in males and females, slightly predominating in the former, and although the valvular involvement may and probably does often begin in early childhood—the diagnosis is usually made at or beyond middle age. Syphilis plays little if any role. Arteriosclerosis must be considered as a causative agent, particularly in older patients. However, as it will be pointed out later, it is often difficult to differentiate between arteriosclerosis and primary inflammation (endocarditis) as the cause of aortic stenosis. Calcification probably represents both a late healing process and the sclerosing calicification of advancing years. The early lesion of aortic stenosis may be thought of as paralleling that of rheumatic mitral stenosis, the calcium deposits occurring in both valves late in the life cycle of the disease.

Prognosis

The outlook is always precarious. Sudden death as in angina pectoris may be the first and last episode. Various writers comment on the long duration of compensation in aortic stenosis, contrasted with the short duration of life once heart failure has

SCIENTIFIC MEETINGS

The Fifteenth Scientific Meetings of the American Heart Association will be held at Hotel Jefferson, St. Louis, on May 12 and 13, 1939. The general cardiac program will be given on Friday, May 12, and the program of the Section for the Study of the Peripheral Circulation on Saturday, May 13. Make your hotel reservations now!

set in. Bronchopneumonia, pulmonary infarction, acute pulmonary edema are often terminal. Early isolated left ventricular failure with gallop rhythm an alternation of the pulse, eventually gives way to systemic venous engorgement with signs of congestive heart failure.

Electrocardiography

Left ventricular preponderance is an almost universal finding in aortic stenosis. Disturbances in auriculoventricular and intraventricular conduction, bundle-branch block, delayed P-R interval, complete heart block are fairly common (Contratto and Levine). These authors comment on the frequent association of conduction defects and aortic stenosis in patients suffering with angina pectoris. Boas believes extension of the calcific process into the ventricular septum may be a factor in these conduction defects, as well as extension of a secondary infection of the diseased valve into the auriculoventricular bundle. Auricular fibrillation and extrasystoles from various foci have been described by several authors. The blood pressure is not particularly significant. Levine comments—"when no other mechanisms are involved the systolic pressure is likely to be low and the diastolic slightly elevated, resulting in a small pulse pressure."

Own Series

We have selected from the files of Michael Reese Hospital 34 cases of aortic stenosis which have come to autopsy. The youngest was eleven years of age, the oldest 82. Cases are found in all intervening decades—the greatest number—11 occurring in the 6th decade. Death at this time coincides with the view that patients with aortic stenosis maintain compensation until a late period in life—until or beyond middle age.

Nineteen males and 15 females comprise the 34 patients. It is interesting to note that in the first 3 decades of life—females predominate 4 to 3—while in the 6th decade—the period of greatest mortality, males predominate 8 to 3. Inasmuch as the diagnosis of aortic stenosis is most frequently made in the later years of life, these comparative figures explain the apparent greater frequency in males. They also suggest that males with aortic stenosis have a better chance of survival than females.

The clinical diagnoses of these 34 cases of aortic stenosis are interesting and clearly portray the difficulty of correct bedside diagnosis. Likewise, they suggest the frequency of combined or dual etiologic factors. The clinical diagnoses were as follows: rheumatic heart disease—12, arteriosclerotic heart disease—9, syphilitic heart disease—3, subacute bacterial endocarditis—1, congestive heart disease—1, no cardiac diagnosis—8. These latter included a brain tumor, agranulocytic angina, acute pancreatitis, diabetes mellitus, uremia, carcinoma of the prostate, carcinoma of the esophagus, and tabes dorsalis. Death in these 8 patients from disease other than the heart substantiates the view that individuals with aortic stenosis frequently carry on for many years, dying eventually of an independent disease. Of those patients in which a cardiac involvement was suspected, the largest number—12—were considered to have rhuematic heart disease, although they comprise only about ⅓ of the total.

In contrast to these clinical diagnoses, at autopsy 15 of the 34 cases were considered to be of the arteriosclerotic type, 11 rheumatic, 5 non-specific in origin. These results are somewhat in conflict with the present generally accepted view of the rheumatic etiology of aortic stenosis. They suggest that either additional etiologic factors are active in aortic stenosis or that the underlying rheumatic process cannot always be recognized, even in the pathological laboratory, or that finally, aortic stenosis may be caused by agents other than acute rheumatic fever.

Because of the discrepancy in opinion regarding the cause of aortic stenosis, and because of the high proportion of cases suggestive of the arteriosclerotic nature of aortic stenosis in our series, it is deemed important to show upon what anatomical evidence the diagnosis of arteriosclerotic stenosis and of stenosis the result of an old (rheumatic) endocarditis was based.

Concerning old rheumatic endocarditis as the underlying cause for the valvular deformity, it must be emphasized that this diagnosis is made not only by the demonstration of actual inflammatory changes in the valve, such as adhesions between the lateral parts of the aortic cusps, but mainly by the microscopic changes in the myocardium. Either perivascular areas of fibrosis were found with remnants of cellular elements which could be interpreted as evidence of old Aschoff bodies, or outspoken Aschoff bodies were demonstrated. If none of these changes were present, and the valve lesions were unquestionably inflammatory, the endocarditis and resulting stenosis of the aortic orifice were designated as "nonspecific" in origin though it is readily admitted that the valvular changes might have been caused by rheumatic fever.

Grossly the cusps of the aortic valve were thickened, rigid and often calcareous plaques were present at the base of the Sinus of Valsalva. Whereas the body of the aortic cusps often was rigid and "stony," the line of closure invariably remained tender and delicate. It must be emphasized that in arteriosclerosis the stenosis of the aortic orifice is the result of calcareous deposits and of the rigidity, while stenosis the result of primary inflammation is usually caused by adhesions between the lateral parts of the cusps. The presence of severe coronary sclerosis with fibrosis or infarcts may also be taken as additional evidence of the arteriosclerotic nature of the valve lesion.

Although there seems to be a growing tendency to minimize arteriosclerosis as cause of aortic stenosis, we believe that the evidence brought forward indicates that in our series arteriosclerosis was the causative agent 15 times.

Of the 34 cases, 15 showed aortic stenosis alone, 19 combined aortic stenosis and regurgitation. This latter figure suggests the necessity of considering carefully the presence of aortic stenosis in each case of clinically diagnosed aortic insufficiency and vice versa.

The average weight of the heart was 505 gms.—the largest 1100 gms. occurring in a boy of 15 years of age with combined aortic insufficiency and mitral stenosis and insufficiency. The smallest heart—300 gms.—was found in an aged man 76 dying of ascending pyelonephritis and uremia.

There were only 4 patients who died suddenly. The predominant cause of death in 17 patients was advanced congestive heart failure. Two patients died of superior mesentery artery and cerebral artery emboli respectively—2 of coronary thrombosis. The infrequency of sudden death in this series is probably to be explained by the character of our material—that of a general hospital—as contrasted with instances of sudden death occurring outside hospital walls.

The frequency of involvement of the other heart valves in our series of aortic stenosis is interesting and suggestive. Except for a congenital bicuspid aortic valve with superimposed subacute bacterial endocarditis in a 21-year-old boy, all the remaining 33 cases showed other valves affected as well. Twelve patients had both mitral and aortic stenosis; 8 mitral, tricuspid and aortic disease; 2 mitral, tricuspid, pulmonic and aortic disease. One must conclude therefore, that rheumatic aortic stenosis seldom occurs alone, that if sought for, other valves will be found affected, that probably the frequency of involvement of other valves is an additional factor in explaining the infrequency of the diagnosis of aortic stenosis.

A sad commentary on the diagnostic skill and acumen exhibited in our series of proved aortic stenosis is demonstrated by the infrequency of the clinical diagnosis. In our 34 cases only once was the diagnosis of aortic stenosis clearly stated. Aortitis was mistakenly diagnosed 4 times, thrombo-ulcerative endocarditis of the aortic valve once. Perhaps, with a review such at this, and similar ones calling attention to the frequency and significance of aortic stenosis, increasing clinical recognition and understanding will be reflected.

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